

Combining Ability of field resistance in cassava (*Manihot Esculenta Crant*) to the cassava Anthracnose disease in Nigeria

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ABSTRACT

A design II making scheme of line and testers involving 4 improved clones as females and 9 landraces were used to produce 36 F1 hybrids of cassava *Manihot esculenta*. The parents and their hybrids were evaluated for their reaction to cassava Anthracnose disease (CAD) at 12 MPM (months after planting) under natural infection in Ibadan in 1998. This was to estimate heterosis, determine the relative importance of general combining ability (GCA) and specific combining ability (SCA) and compare line and topcross for resistance to CAD.

Results showed that genetic variation was due predominantly to the GCA of female. Eleven crosses had significant negative heterosis. All the improved lines except 130572 contributed significantly to GCA effect for resistance. TME 3; TME 6 and TME 11 were best male general combiners. Parental performance can be used to predict progeny performance because the line performance was positively correlated with topcross performance.

Key words:- Combining ability, heterosis, resistance, cassava, Cassava Anthracnose Disease.

INTRODUCTION

Cassava anthracnose disease (CAD) is the most important fungal disease of cassava in the field in West Africa especially Nigeria (Hahn *et al.*, 1989). In recent years, CAD has become so important in the extent of damage to cassava from about 2 months to 12 months after planting (MAP) plants. This is by reducing the amount of healthy plantable items available to the farmers. Hence, a search for cultivars of cassava resistant to CAD is relevant. According to Hahn *et al.* (1989) young cassava stems of about 3 – 4 MAP attacked by CAD is characterized by oval, pale brown, shallow depressions, which could lead to petiole epinasty, necrosis, wilting and defoliation (Muyolo, 1984). Van der Bruggen (1987) observed that infection on older cassava plants of 7 – 12 MAP usually cause round stringy lesions that develop into deep cankers, causing stem to become brittle and easy to break by wind action. It has been observed that chemical control and roughing of all infected plants are not feasible since major disease such as African Cassava Mosaic Virus (ACMV), Cassava Bacteria Blight (CBB) and CAD are already wide spread and their methods of dissemination complicated. The only most effective means of control is by cultivating resistance varieties (Lazano, 1989). Breeding for resistance to disease aims at improving cultivars'

resistance in a wide range of environmental conditions for a long period (Jennings, 1976).

Some African landraces have been identified to be resistant to CAD, which could serve as a new source of resistance to the disease. When introgressed into improved cultivars, new resistant genotypes could be developed. To ensure that durable resistance is maintained, additional sources of resistance with a wider genetic base are sought to diversify resistance to the disease that will prove difficult for the pathogen to overcome. However, selection of parents to be in a breeding program cannot be based on their performance per se. Knowledge of the relative importance of general combining ability (GCA) and specific ability (SCA) are essential in formulating an efficient breeding strategy. When two or more parents are hybridized to develop a new line or cultivar, it is important to know the combining ability of the parent so as to make use of phenomenon of epistasis (Hayman, 1958).

Therefore, in this study 36 F1 crosses and their parents, developed in a design II mating system (Mather and Jinks, 1982; Singh and Chaudury 1985) were evaluated in 1997 and 1998 seasons to determine the relative importance of GCA, and SCA, estimate heterosis and correlate line and topcross performance for resistance to CAD.

MATERIALS AND METHODS

The experimental materials included four International Institute of Tropical Agriculture (IITA) improved cassava clones (females) and nine African cassava landraces (males) and their F1 progenies developed by design II mating system at IITA research field in Ubiaja, Edo State, Nigeria in 1996. The design II mating scheme (Mather 1974, Mather and Jinks 1982) ensures random mating by using a common male (n_1) and a common female (n_2) to produce progeny families with $\frac{1}{2}$ sib relationship. The total variation among the resultant progenies belonging to n_1 n_2 families were partitioned to the various sources to explain line \times Tester analysis (Topcross) (Table 1). These parents and their 36 progenies were evaluated under rain-fed conditions in 1997 and 1998 for their susceptibility to CAD.

Matured stakes (25 cm long) of both the parents and their hybrids were planted. The hybrids were planted in a serpentine row trial augmented design (Fasoulas 1973). This is to enhance individual observation for number of hybrids involved. It involved 36 F1 crosses and the parents were planted in a randomized complete block design with two replications. The ridges were spaced 1m apart, 30 cm high and 0.75 m wide. No fertilizer or herbicide was applied during the period of the study. Hand weeding was done when necessary.

The plants were monitored from 2 – 12 MAP for CAD symptoms under natural disease conditions in an area known for CAD epidemics. Each plant was evaluated for symptoms of CAD and data were collected and scored using the method adopted by Ikotun and Hahn (1991) as follows.

- (1) Distance of cankers from the ground level (cm)
- (2) Total number of cankers/plants (visual counting)
- (3) Size of the largest canker on matured stems (mm).
- (4) Size of the largest canker on young stems/shoots (mm).

STATISTICAL ANALYSIS

General combining ability (GCA) procedure (SAS, 1996) was used to calculate the average disease scores of the parents and F1 hybrids [where 1 = Resistant (R), 2 = Moderately Resistant (MR) and 3 = Tolerant (T). The sum of squares of genotypes was partitioned into variations due to parents, crosses and parent vs. cross. Parents were further partitioned into variations due to males, females and interaction between female parent and male parent.

Variance components of random effects were calculated using Mather and Jinks (1982), Singh and Chaudhary (1985), Griffing (1956). Mid parent heterosis of hybrids were obtained as follows:

$$\text{Heterosis: } \frac{\bar{X} \text{ hybrid} - \frac{(\bar{X} \text{ Male} + \bar{X} \text{ Female})}{2}}{\frac{\bar{X} \text{ Male} + \bar{X} \text{ Female}}{2}}$$

where:

\bar{X} = means of male, female and hybrids respectively.

Combining ability analysis was calculated using the methods described by Beil and Atkins (1967). Simple phenotype correlations using line and top cross means were obtained to compare topcross (line \times tester) performance of parents (Falconer 1967).

RESULTS AND DISCUSSION

Results of the analysis of variance (Table 1) showed that there were significant variations amongst crosses for resistance to CAD at 12 months after planting (MAP) and that females contributed more to the variation of CAD reactions. Orthogonal contrast of female versus male, were significant. The significance of parent contrasts are indications of diverse variability amongst the parents used in this study. The parent also differed significantly from their crosses for resistance to CAD.

The significant variation among the crosses was predominantly due to GCA effects of the female parents. Only negative values of GCA and SCA indicated contributions toward resistance. Highest negative GCA effects for resistance to CAD was observed for female parents TME 3, TME 6, and TME 117 (Table 3). This is an indication that they are best general combiners. Male parents TME 4, TME 9, TME 11 and TME 12 were intermediate general combiners. The female parent 130572 and male parent TME 7 were poor general combiners. Significant negative SCA effects were observed among the crosses for resistance to CAD. At least 14 superior specific combiners were observed with significant negative SCA effects. 130555 \times TME 117 was the best specific combiner among the crosses, whereas 130572 \times TME 117 was the poorest specific combiner. However, the cross between two moderate susceptible good general combiners, 130555 \times TME 117 had the highest significant SCA effects for resistance to CAD. Moreover, the cross between two resistant good general combiners 130001 \times TME 4 had significant negative SCA effect. It therefore can be inferred from Table 3 that there were additive and epistatic gene effect. This is because high and significant negative GCA and SCA values were recorded in the resistance of the parents and the hybrids to CAD. This agrees with the work of Malek (1976) on combining ability of grains per spike in diallel crosses of six wheat cultivars. Jinks (1955) and Falconer (1967) had earlier used combining ability

methods on wheat grains. In addition, the GCA variance component used due to female was 364.36 while that due to male was 131.00 and SCA was 25.68 (Table 4). The ratio (GCA/GCA + SCA) was 0.96 indicating that GCA was more important in predicting progeny performance. This is in consonance with the studies of Jennings (1976) and Manners (1993). The orthogonal contrast, parent versus cross was significant which is an indication of heterosis amongst the crosses. Eleven crosses had significant negative heterotic effect ranging from -0.02 to -0.83. The highest positive heterotic value of 3.20 was obtained for 130572 × TME 117 (Table 5). The occurrence of negative values for heterosis when the F1 means is less than that of the better parent in a cross is desirable for resistance to CAD (IITA, 1990). The significant negative value of heterotic effect in the cross 130572 × TME 11 suggest the presence of

positive resistance factors even in the susceptible parents. However, heterosis would occur when there is a difference in gene frequency. The results of this investigation revealed that the parents had variability for resistance to CAD. The significant levels of heterosis may be due to these variations.

The performance of parents was positively correlated with their topcross ($r = 0.32^*$) indicating that progeny performance could be predicted based on parent's performances. The relative magnitude of $GCA/GCA + SCA$ and $2r^2g/2r^2g + r^2$ suggest the reliability of GCA for determining progeny performance.

where

r^2 = Coefficient of determination

r = Correlation coefficient

g = General combining ability for each cross:

Table 1. ANOVA for CAD canker counts of 4 improved parent (females), 9 landraces (male) and their hybrids evaluated in Ibadan 1998 and 1999. (Design II mating scheme).

Source	df	Mean square
Rep	1	64.33
Genotypes	48	910.69**
Parents (P)	12	209.82**
Crosses (C)	35	1160.37**
Female (F)	3	1643.09**
Males (M)	8	296.83
F × M	24	779.4*
PvC	1	843.53**
Error	48	165.53

* ** Significant at 5 and 1 % respectively

Table 2. Means CAD canker counts of crosses of 4 improved and 9 landraces

Female	Male								
	TME 11	TME 117	TME 12	TME 3	TME 4	TME 6	TME 7	TME 8	TME 9
130001	15.11	12.41	8.20	10.22	8.31	5.86	8.05	6.83	8.35
130055	6.06	5.0	9.75	8.67	9.88	10.32	10.50	16.81	10.30
130572	10.28	41.16	17.27	10.18	11.93	8.10	8.56	12.77	11.95
163397	8.94	8.46	6.46	5.88	13.00	6.90	1.60	9.50	8.96

Table 3. Estimate of general and specific combining ability effect for resistance to CAD

	130001	130555	130572	163397	gi effects of male
Male	Specific effect				
TME 11	6.09	-3.82	-4.43	1.49	0.26
TME 117	-3.30	-10.88	19.85	-5.68	0.01
TME 12	1.17	1.75	3.21	1.34	0.86
TME 3	2.89	0.67	3.14	0.38	1.48
TME 4	-1.42	-0.01	3.39	6.74	0.42
TME 6	0.89	3.40	-4.26	1.72	-2.56
TME 7	1.92	-7.74	-1.21	-2.86	3.09
TME 8	-3.61	6.19	-3.26	0.67	1.13
TME 9	-0.48	1.30	2.8	1.70	0.48
gi effects of females	-1.86	-0.85	-4.54	-2.62	

Table 4: Components of variance

Components	Total number of cankers/plant
$\sigma^2_{g_j}$	364.36
$\sigma^2_{g_i}$	131.00
$\sigma^2_{s_i}$	25.68
$2\sigma^2_{g/} / (2\sigma^2_{g/} + \sigma^2_{s/})$	0.96

 $\sigma^2_{g_j}$ = variance component of the female $\sigma^2_{g_i}$ = variance component of the male $\sigma^2_{s_i}$ = variance component of the specific combining ability

Table 5. Heterotic effect for resistance to CAD

Hybrid	Mean value of parent 1	Mean value of parent 2	Heterotic effect
130001 × TME 11	3.35	17.35	0.45
130001 × TME 117	3.35	8.07	1.17
130001 × TME 12	3.35	6.08	0.74
130001 × TME 3	3.35	6.85	0.72
130001 × TME 4	3.35	6.06	0.77
130001 × TME 6	3.35	7.08	-0.15
130001 × TME 7	3.35	10.73	0.15
130001 × TME 8	3.35	8.94	0.11
130001 × TME 9	3.35	7.84	0.49
130555 × TME 11	8.50	17.35	-0.11
130555 × TME 117	8.50	8.07	-0.48
130555 × TME 12	8.50	6.08	-0.16
130555 × TME 4	8.50	6.85	0.22
130555 × TME 6	8.50	6.06	0.32
130555 × TME 7	8.50	7.08	0.33
130555 × TME 8	8.50	10.75	0.93
130555 × TME 9	8.50	8.07	0.26
130572 × TME 11	11.24	9.1	-0.29
130572 × TME 117	11.24	7.84	3.20
130572 × TME 12	11.24	17.35	1.00
130572 × TME 4	11.24	8.07	0.13
130572 × TME 6	11.24	6.08	0.37
130572 × TME 7	11.24	6.85	-0.22
130572 × TME 8	11.24	6.06	0.07
130572 × TME 9	11.24	7.08	0.26
163397 × TME 11	11.24	7.84	-0.32
163397 × TME 117	11.24	17.35	-0.02
163397 × TME 12	9.28	8.07	-0.16
163397 × TME 3	9.28	6.08	0.27
163397 × TME 4	9.28	6.85	0.36
163397 × TME 6	9.28	6.06	-0.15
163397 × TME 7	9.28	7.08	-0.83
163397 × TME 8	9.28	10.73	0.05
163397 × TME 9	9.28	8.94	0.32

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